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AMERICAN JOURNAL OF OPTOMETRY

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AN OPTOMETRIC MEANS FOR THE CONTROL OF PRIMARY GLAUCOMA*

Charles I. Saks, D.O.S.
Syracuse, N. Y.

Primary glaucoma, or hardening of the eyeball, is one of the most insidious eye diseases known. It usually occurs after the age of fifty and is very common to the Jewish race, being most common in women.[100] In a home for the aged blind the percentage of blindness due to glaucoma was 29.82%.[101]

It will be shown that evidence contradicts the commonly accepted theories on the mechanism and causation of primary glaucoma.

The extremely doubtful therapeutic value of drugs in the treatment of primary glaucoma will be revealed by means of clinical evidence and opinions derived from highly authentic sources.

I will show that the presence of preglaucoma may now be determined, and that the prevention and control of primary glaucoma now appears to be an actuality. I will further show that the intraocular tension appears to have a definite relationship to the occurrence of nocturnal and early morning heart attacks.

Miosis is thought by many to be the factor in the relief of glaucoma. While eserine or pilocarpine cause miosis, it does not seem likely that it can cause relief of increased intraocular pressure.

The pupil contracts in sleep and yet the statistical intraocular pressure is greatest in the normal or glaucomatous eye in the early morning hours.[1, 9] If tension is relieved by means of eserine or pilocarpine, it may be due, according to Duke-Elder to contraction of the ciliary muscle which exerts a three fold influence,[2] by diminishing the blood supply to

*An abridgement of the material presented before the Inter-Professional Relations Committee of the New York State Optometric Society at New York City, April 30, 1939. Submitted for publication May 8, 1939.

the anterior portion of the uveal tract by constricting the anterior arteries as they pass through the muscle belly; by opening up the choroidal veins and the canal of Schlemm by traction on the scleral spur.

However, pilocarpine and eserine cause dilation of the intraocular vessels,[4] and it will be shown later that the therapeutic value of miotics in the treatment of primary glaucoma is very questionable.

It would seem that accommodation of itself would cause an increased tension because there is positive experimental evidence to prove that traction on the choroid during accommodation causes the vitreous to be advanced. Enlarged photomicrographs of monkeys' eyes fixed after instillations of eserine and atropine show that the vitreous is pressed forward against the periphery of the lens, the zonule and ciliary body under eserine; while an open space exists between these structures in atropinized eyes.[3]

Actual practice reveals that accommodation does not appear to cause increased tension, for it was shown that the statisticial tension is greatest in the early morning hours.[1] Adler has shown that use of the eyes in glaucoma does not cause the tension to increase,[30] and statistics show that age or the refractive condition have no effect on intraocular pressure.[1] If accommodation caused increased tension, then presbyopes and myopes would have less tension than hyperopes and emmetropes of younger years. Furthermore, Luedde has shown that increased intraocular tension is not observed during accommodation because there is merely a rearrangement, and not an increase of the intraocular contents.[3] According to Grönholm,[93] contraction of the ciliary muscle in the experimental animal by stimulation of the third nerve causes no change of the intraocular pressure.

According to Duke-Elder, pilocarpine, eserine and atropine all cause dilation of the intraocular capillaries, increasing their permeability, and thereby increasing the fluid content of the eyeball.[4] However, pilocarpine and eserine at the same time cause a contraction of the ciliary muscle compressing the anterior arteries as they pass through the latter muscle belly. Atropine relaxes the ciliary muscle probably allowing more blood to enter the anterior portion of the uveal tract.

Either eserine[28] or atropine[91] causes a rise in pressure in the normal eye. According to Duke-Elder[28] this is caused partly by dilation of the minute intraocular vessels and increased permeability of their walls.

Eserine,[28] according to Duke-Elder, also causes increased tonicity of the recti.

While a tenotomy of the recti lowers the pressure (Schönberg, 1913, Salvati, 1924),[29] and the fact that pressure of the eye falls to almost half of its original height when the recti are paralyzed by curare,[29] and stimulation of the recti to contraction by acetyl-choline causes increased pressure, we must consider that medical or surgical interference of the recti is not a physiological condition. The recti and obliques cannot be considered as causating factors of glacoma because the statistical pressure in the normal or glaucomatous eye is highest in the early morning hours.[1] Furthermore, Adler has shown that use of the eyes in glaucoma does not cause the tension to increase.[30]

The effect of light on the intraocular pressure introduces a factor that cannot be overestimated in importance. Feigenbaum (1928-29) showed that in dark adaptation the intraocular vessels dilate and that the tension rises.[5] He found this to be so in blind as well as seeing eyes, occurring indifferently in aniridia, after an iridectomy, and with atropine or pilocarpine. It has also been shown that the intraocular capillaries constrict on exposure to light causing a drop in pressure.[6]

Since the statistical tension is greatest in the early morning hours,[1] it seems very likely that this increased tension is due to sleeping in the dark.

According to Gradle,[7] of glaucomatous patients kept in a very dark room for one hour, more than 50% will show the tonometric findings at the end of this period to be increased from 15 to 40 mm. Hg. above the reading taken before being placed in the dark room. Upon exposure to strong light, there follows a *sudden* drop in tension to the *previous* level. This fact may explain why the eye specialist seldom sees a true prodromal attack. Quote: "The ophthalmologist[43] seldom sees a true prodromal attack and therefore must diagnose the condition upon the history given by the patient, for after subsidence, the eye may present no evidence of hypertension." Unquote: This is probably because the patient consults the specialist during the day after light adaptation has brought the tension down to normal.

It is to be noted that the statistical data reveal that tension is highest in the early morning hours. The fact that exposure to darkness of glaucomatous patients for one hour[7] causes a rise in tension of 15 to 40 mm.

Hg. in more than 50% of paients submitted to this test, the fact that Parsons has shown that attacks of acute glaucoma come on mostly at night,[23] and the fact that Alger has shown that cases of chronic simple glaucoma reveal abnormally high tension only during the night,[33] makes it seem quite evident that tension is in truth highest during the night. Hence the pressure in the early morning hours shall be referred to as the statistical pressure. It will be shown later that the hour of maximum intraocular tension depends on the time that the patient retires.

The fact that exposure to light after being submitted to darkness causes a sudden drop in tension to the previous level appears to prove that it is the actual dilation of the intraocular vessels[5, 6, 23, 33, 38, 39, 94, 95] and not the increased dialyzed fluid content (thought to be caused by the increased permeability of the dilated vessel walls)[38] that causes the increased pressure. If the increased tension were actually due to the increased dialyzed fluid content, exposure to light would cause only a partial drop in pressure. This fact also seems to show that the active factor in the causation of primary glaucoma cannot be interference of drainage.

According to Adler[38] change in the intraocular pressure from contraction or dilation of the distensible blood vessels of the eye is chiefly of the choroidal circulation. The changes in intraocular pressure are not due to any interposed mechanism such as increased fluid formation.

The choroid consists chiefly of layers of blood vessels so that the histologic appearance of its cross section resembles that of spongy tissue. The state of contraction or dilatation of these blood vessels, particularly of the chorio-capillaris, will determine the volume of blood in the interior of the eye and hence the intraocular pressure.[38]

According to Wolff[39] the chorio-capillaris consists of capillaries of wide bore, closely packed together, are much wider than elsewhere and show many sac-like dilatations. They form a net whose meshes are especially close and regular behind at the fovea centralis, and receive, according to Hepburn, a special arterial twig, while anteriorly they become wider and wider, being remarkably wide at the ora serrata. The arteries show a well developed muscularis with an adventitia made up of almost homogeneous collagenous tissue containing thick elastic fibres. The capillaries of the chorio-capillaris are characterized by their size. Whereas in an ordinary capillary there is hardly room for one red blood corpuscle to pass at a time, here there is room for several.

Gradle claims the increased tension of those submitted to the dark room test is due to partial obstruction of the angle of the anterior chamber caused by dilation of the pupil. As was previously shown the size of the pupil does not seem to affect the intraocular pressure because statistics show that it is greatest in the early morning hours even though the pupil contracts in sleep.[1,9] An atropinized pupil will also contract considerably in sleep.[8,9] Furthermore, if dilation of the pupil causes the increased tension, why isn't there an increased tension in all the cases submitted to the dark room test? It is to be noted that in normal eyes, the tension bears no relationship to the size of the pupil, for many people with large pupils have no more tension than those with small pupils.

As was previously shown, dark adaptation causes dilation of the intraocular vessels resulting in increased tension in blind and seeing eyes, occurring indifferently in aniridia, after an iridectomy and with pilocarpine or atropine.[5]

According to Gradle[7] only slightly more than 50% of glaucomatous eyes submitted to the dark room test show an increased tension. One reason that the other 40 and some odd per cent do not show this increase is probably because of a loss of tone of the capillary and arteriole walls, or because of arteriosclerosis involving the intraocular vessels. Another and perhaps the chief reason is the lag of dark adaptation, for it was shown by Parsons[23] that attacks of acute glaucoma occur especially at night, and Alger[33] has shown that after the onset of presbyopia in middle life, chronic simple glaucoma reveals abnormally high tension only during the night. In other words, it seems evident, as will be shown later, that all glaucomatous patients will reveal a rise in tension after being submitted to darkness a sufficiently long time. It will also be shown later that the anomalous behavior of the regenerated visual purple in dark adaptation appears to be the basic cause of increased intraocular tension.

Sclerosed vessels have a reduced lumen (arteriosclerosis causes a thickening of the walls inwardly thereby reducing the lumen of the vessels)[35] and the vessels so affected will be able to dilate to a very limited extent, if at all, depending on the extent of the arteriosclerosis.[10]

Arteriosclerosis is not to be assumed to mean the same as high blood pressure. It is established that there may be arteriosclerosis without high blood pressure at any stage, and high blood pressure without arteriosclerosis.[11]

The fact that dilation of the intraocular vessels in dark adaptation causes increased tension even in normal eyes may explain occasional ocular pains and headaches in movies or when arising from sleep in the morning when there is nothing else to account for the ocular pains or cephalalgia. This point is borne out vividly by actual results in patients both young and old in my own practice. By sleeping with a lighted five to seven watt bulb over the face, morning headaches, dizziness and nausea were either completely or partially relieved in a number of cases. The reason most people are not similarly affected is probably because darkness does not cause dilation of the vessels to any marked extent. This depends on the ability of the intraocular vessel walls to dilate, and the behavior of the light and dark adaptive functions of the retina.

Since drugs and surgical interference do not insure against further attacks of glaucoma,[34] and the fact that exposure to darkness causes increased tension even with pilocarpine or after an iridectomy,[5] emphasizes very strongly that light and darkness supercede the effects of drugs and surgery.

Luckiesh and Moss[18] found in normal healthy subjects a progressive decrease in the heart rate as the duration of the visual task was increased, and that the decrement in the heart rate was much greater under one foot candle than it was under 100 foot candles. The decrease in the heart rate while reading one hour under one foot candle is 10%, whereas the decrease for a similar period under 100 foot candles is only 2%.

This phenomenon is probably due to the oculocardiac reflex.[19] Through an association of the trigeminal and vagus nerves increased pressure in or on the eyeball causes a slowing of the heart rate. In the normal state more blood flows to the eyes in the process of reading causing dilation of the intraocular vessels probably resulting in a slightly increased tension. As illumination is reduced, the intraocular vessels will dilate further allowing more blood to enter the eyeball, thereby further increasing the tension[5] and resulting in a further slowing of the heart rate.

It is questionable if reading would cause the oculocardiac reflex in cases of arteriosclerosis of the intraocular vessels to the same extent as in those cases where no arteriosclerosis exists because of their rigidity and reduced lumen.[10, 35] The effect on the heart rate of different levels of illumination also depends on the dark-adaptive properties of the retina. This point will be discussed under the etiology of primary glaucoma.

While Adler[30] has shown that use of the eyes in glaucoma does not cause the tension to increase, the reading test under variable levels of illumination may reveal an effect on the heart rate in glaucomatous patients, especially under low illumination.

It is also doubtful if the oculocardiac reflex in people with one eye would cause as much slowing of the heart rate as in people having two eyes. In one-eyed people (those having only one eye) the stimulus causing the oculocardiac reflex would not be as intense as in people having two eyes.

Having two eyes with only monocular vision is not to be thought of as being similar to having only one eye, for, when one eye only is exposed to light, the reflex travels to the other eye also, the conduction being stopped by an optico-ciliary neurectomy (Feigenbaum, 1928-29).[21]

To show that the state of dilation or constriction of the intraocular vessels is individual from and opposite to that of the occipital cortex, the following case is cited: Fulton[89] reports a case of a man with a circumscribed racemose arterial angioma of the occipital cortex in whom a distinct bruit could be heard over the site of the lesion after the skull had been trephined. The tumor could not be removd so the trephine opening was left over the tumor! When the patient was at rest with his eyes closed, the bruit was distant and weak, but when he was requested to read, there was a marked increase in the bruit, detectable both by ausculation and electro-phonograms. This increase in the intensity of the bruit occurred within twenty to thirty seconds after the patient began to use his eyes, and persisted for nearly a minute after the light was turned off. We may imply from the latter statement that the bruit was again distant and weak when remaining in darkness, or in other words, the vascularity of the occipital cortex diminished. This is admitted by Fulton who concluded that visual effort was associated with an increase in the vascularity of the occipital cortex. We know that darkness causes a *dilation* of the intraocular vessels resulting in their *increased* vascularization.[5, 6, 7, 23, 33, 38, 39, 94, 95]

Grossman[32] found that in cases of aniridia, the diameter of the lens under atropine is 12.25 mm.; under eserine 10.25 mm.; and in the resting state 11.50 mm. Some may therefore conclude that relaxation of accommodation during sleep may cause increased tension. This point may be ruled out because it has been shown that the average intraocular tension in myopes and presbyopes is no greater than in younger hyperopes.[1]

The association of the trigeminal and vagus nerves is further revealed in the nausea experienced in cases of uncorrected refractive errors and in cases where the Rx is causing ocular discomfort. In these cases there probably will be increased vascularization of the intraocular tissues. A more marked example of the effect of increased intraocular tension is vomiting in some cases of glaucoma.

Here we have an example of the "all or none" theory.[20, 62] In uncorrected ametropia or where the Rx causes ocular discomfort, the intraocular tension is probably increased slightly, merely causing nausea. Where the tension is increased markedly as in glaucoma, more receptors of the trigeminal are stimulated causing a more intense stimulus resulting in vomiting.

The following quotation from Evans[62] is self-explanatory. Quote: "As a final objection to any theory which assumes that different portions of a zone can transmit different degrees of intensity over the single connecting fiber, it may be said that all recent work on sensory receptors has shown that they obey the all-or-none law similar to the nerve impulse in a motor fiber. There is every reason therefore to believe that fractional stimulation of a cone or even fractional conduction in a nerve fiber is impossible."

An iridectomy may probably cause relief from glaucoma because:

1. The rate of aqueous secretion may be reduced because some portion of the intraocular capillaries is removed for it is thought by some that the iris contributes to the formation of aqueous.[82, 83]

2. According to Parsons[22] it may be probable that additional drainage is absorbed by the cut edges of the coloboma, for wounds of the iris do not heal by cicatrization, the lymph spaces of the iris stroma always remaining continuous with the anterior chamber at the cut margins.

However dark adaptation as was brought out previously will cause increased tension even after an iridectomy.[5]

Seefelder[36] found that out of 82 cases of congenital aniridia, 23 had glaucoma. However, the anomalous iris root, often found to be present in these cases by histologic examination,[36] was found to be usually directly adherent to the posterior surface of the cornea, thus blocking the angle of the anterior chamber.[37]

The fact that dark adaptation causes a rise in tension primarily by dilation of the intraocular vessels in cases of aniridia or where an iridectomy was performed, with atropine or pilocarpine,[5] makes it seem quite evident therefor that interference of drainage is a very doubtful cause of primary glaucoma. Drainage of the eye occurs at a very slow rate in normal eyes[83] and there is no reason why it should be diminished in dark adaptation.

Histologic evidence reveals that the individual fibres of the pectinate ligament are somewhat thickened and sclerosed so that the character of the filter is changed and the total filtering area supposedly lessened.[12] According to Friedenwald[31] many cases of glaucoma are seen where no such lesion is found. He states further that there is no evidence that sclerosis of the pectinate ligaments interferes in any way with the movement of the fluid in the eye.

To show further that it is dilation of the intraocular vessels and not interference of drainage that appears to cause increased tension, the following reasons are presented:

1. The *sudden* drop in pressure of glaucomatous eyes to the *previous level* when coming from darkness to light.[7] If the increased tension were due to interference of drainage, exposure to light would cause only a partial drop in pressure.

2. According to Parsons, attacks of acute glaucoma come on especially at night.[23]

3. Alger has shown[33] that, after the onset of presbyopia in middle life, chronic simple glaucoma reveals abnormally high tension only during the night.

4. According to Adler,[38] Feigenbaum,[5,6] Hamburger,[94] and Friedenwald,[95] the changes in intraocular pressure are due to the contraction and dilation of the intraocular vessels.

It appears that senile changes responsible for the causation of primary glaucoma are found in the circulatory and visual purple forming elements of the retina. Senile changes of the choroid do not appear to be concerned with the causation of primary glaucoma.

As age advances there is a deposit of connective tissue in the uveal tract,[13,14,15,16] increasing considerably with age. This deposit of connective tissue appears to involve the stroma of the choroid merely affecting its elasticity. This appears to be evident because the accommodative

amplitude decreases with age and a higher reading addition is required as presbyopia advances. I have observed that high blood pressure is very often found in those presbyopes requiring a higher reading add than expected for a certain age. [13, 14, 15] Furthermore Clapp [27] found that a drop of homatropine instilled into the eye of a person aged 40 will cause lessening of the accommodation in 10 to 12 minutes, while in the young it will take much longer. The choroid in one of forty is so rigid in comparison with that in one of younger years that the zonular fibres are rendered tense and taut in a shorter time. This fact seems to emphasize that presbyopia is due mainly to a rigidity and loss of elasticity of the choroid. [13, 14, 15]

To show further that the deposit of connective tissue in the choroid merely involves the elasticity of the choroid and not its blood vessels, Alger [33] has shown that after the onset of presbyopia in middle life, chronic simple glaucoma reveals abnormally high tension only during the night. Since there is sufficient evidence to show that increased intraocular tension brought on by darkness is due to a dilation of the choroidal vessels, [5, 6, 7, 38, 39, 23, 33, 94, 95] even in People well beyond the presbyopic age [33] it appears quite evident that the deposit of connective tissue in the uveal tract during senility does not involve the choroidal vessels.

Arteriosclerosis involving the choroidal vessels will resist dilation from whatever cause depending on the extent of the arteriosclerosis. [10] However, it will be shown later that well advanced sclerosis of the retinal arteries that cross retinal veins may give rise to a venous embolism, resulting in primary glaucoma. [48, 49, 50, 52]

The common conception of drainage as a cause of glaucoma may be ruled out. Consider the glaucomatous cases of those presbyopes in the late fifties and beyond who have practically no accommodation. The ciliary muscle in these patients is just as inactive during the day as it is during sleep. Since attacks of acute glaucoma occur especially during the night, [23] and since Alger has shown [33] that after the onset of presbyopia in middle life, chronic simple glaucoma reveals abnormally high tension only during the night, drainage cannot be considered as a factor because of inactivity of accommodation during sleep.

Since glaucoma may occur at any age, and since it occurs mostly in hyperopes, the drainage theory continually loses ground. Many hyperopes up to and in the presbyopic age, have an active accommodation and yet have glaucoma. From these facts, therefore, we may seriously

question the therapeutic value of miotics in the treatment of primary glaucoma. Many myopes do not wear glasses and therefore exert no accommodation when fixating objects beyond their par point. Theoretically myopes should be subject to glaucoma if this disease is thought to be due to interference of drainage.

There may be truly an interference of drainage which is secondary to hypertension. The dilation of the intraocular vessels may cause more or less collapse of the intraocular veins, seriously obstructing venous drainage which will result in edema of the intraocular tissues adding further to the increased hypertension.[63]

Because of the apparent fallacies surrounding the theories of drainage interference through Schlemm's canal as a cause of primary glaucoma, it seems evident that the prevalence of glaucoma among hyperopes is due merely to the fact that our population is made up of a majority of hyperopes.

Furthermore, the fact that treatment with miotics does not assure the arrest of primary glaucoma[34] adds further to the question of doubt concerning the beneficial effects of miotics. In those cases where the progress of glaucoma has been arrested, it is my belief that arteriosclerosis of the intraocular vessels prevented their further dilation to cause sufficient increase in tension.[72]

John N. Evans of Brooklyn, N. Y., presents evidence to show that tension in some cases of glaucoma is *less after the discontinuance of miotics.*[64]

According to Duke-Elder[4] pilocarpine and eserine cause dilation of the intraocular capillaries, increasing their permeability and thereby increasing the fluid content of the eyeball.

According to Gradle[24] cases have been recorded in which an acute rise of tension followed the primary instillation of pilocarpine.

Quote: "Although eserine is by far the more powerful miotic,[24] no case of compensated glaucoma (non-inflammatory) that requires the use of eserine for the maintenance of normal intraocular tension can be treated safely without surgical intervention . . . but experience has shown that continued eserine therapy is definitely detrimental to such an eye. The continued use of eserine frequently causes an enormous hypertrophy of the subconjunctival follicles and a development of a severe conjunctivitis that may temporarily resemble an acute trachoma. Pilocarpine

also causes a conjunctival irritation, but of a much milder form." [17, 26]

Quote: "But even the most favorable cases [25] may show advancement in the course of the compensated glaucoma, as evidenced by changes in visual acuity, visual fields or increased intraocular tension, despite the regular use of miotics."

Quote: "Again, an actual acute attack [42] may expend its violence within a few days, even without active treatment, and may pass into a chronic incompensated (non-inflammatory) form." Unquote: If miotics were essential to the treatment of glaucoma, such untreated cases would probably pass on to blindness."

Quote: "Eyes affected [44] with malignant compensated glaucoma are almost universally doomed to complete blindness within from one to two years."

Quote: "Many cases [34] cannot be controlled by miotics even with the aid of violent reducing measures."

To show further that the pupil does not affect the intraocular tension, I shall quote the following: "If epinephrine is to be used as suggested by Gradle, [25] put upon a small cotton pledget which is tucked into the lower transitional fold . . . within 15 minutes, the pupil will become widely dilated despite the previous use of miotics. The intraocular tension will be reduced by vasomotor contraction of the intraocular vessels. However, there is a certain element of danger in such a procedure; for in some cases instead of falling, the tension rises, due to obstruction of the angle of the anterior chamber by the thickened iris, and an attack of acute incompensated glaucoma results." Unquote: If dilation of the pupil obstructs the angle of the anterior chamber, why does epinephrine reduce the tension in some and increase it in others?

Donald T. Atkinson further reveals the extremely doubtful benefits of miotics in the following quotation. Quote: "Regardless of the favorable effects upon the tension brought about by the use of miotics, the results are only transitory. For this reason it is impossible to cure even the mildest cases of glaucoma by the administration of either eserine or pilocarpine, and their use should never be regarded as more than a temporary measure." [104]

The fallacy of pupillary size and its purported effect on intraocular pressure is well demonstrated in experiments performed by Friedenwald. [95] He produced a rise in intraocular pressure in experimental

animals by injecting histamine into the vitreous. The rise in pressure, which was of very short duration, was followed by a very marked fall. *He found that the time of appearance of the hypotension was materially hastened by dilating the pupil, and that a narrow pupil produced by eserine occasionally prevented the fall of pressure altogether!* This point emphasizes further the questionable value of miotics. It will be noted also that hypotension was materially hastened by dilating the pupil.

According to Dale, 1914; Schenk, 1921; and Hamburger, 1926, histamine [96] is an extremely powerful miotic, acting directly on the muscle fibres, and contracting the pupil maximally even when under the influence of atropine. It is therefore difficult to conceive how Friedenwald dilated the pupil. Furthermore, since histamine contracts the pupil maximally, eserine will be of no further help as far as the pupillary size is concerned.

The term hypertension is a relative one. [41] In cases presenting a tonometric reading of 22 to 24 mm. Hg., the eyes are destroyed by the ravages of glaucoma, whereas cases have been recorded with a pressure of 33 to 38 mm. Hg. which had existed over a period of years without discernible damage to the funtion of the eye. [41] This may be due to the fact that the eye revealing a lower tension during the day may be increased considerably while sleeping in the dark. It is my belief that in those cases revealing 33 to 38 mm. Hg. of tension over a period of years without discernible damage to the function of the eye may be due to the toughness or thickness of the cornea which offers a greater resistance to the plunger of the tonometer.

While Koster and Ridley [79] have shown that the sclera is subject to marked stretching when increasing the intraocular pressure, I do not believe that distensibility of the sclera may be regarded as a factor in the prevention of glaucoma. Some may advance the argument that increased tension causes distension of the sclera in young eyes resulting in myopia. If this were the case then these myopes would suffer the characteristic symptoms of glaucoma before the onset of myopia. Besides it would take a considerable amount of pressure to distend an eyeball. Furthermore statistics show that glaucoma among myopes is by far less frequent than in hyperopes. It will be repeated that statistics reveal the same average tension in hyperopes and myopes regardless of age. [1]

Stretching of the sclera in glaucoma occurs in the form of staphylomata in the region of the equator where the sclera is weakened by the

exit of the vortex veins and where it is unsupported by muscles.[86] Such staphylomata have not been observed in excised myopic eyes.

Stretching of the sclera is observed in infantile glaucoma, or buphthalmos, which is due, according to Wolff to:[87]

1. Congenital defect in which the root of the iris does not become separate from the corneo-sclera because of intrauterine inflammation.

2. Schlemm's canal is absent or defective.

In this condition the eyeball distends all over and usually leads to general enlargement. The sclerotic is thinned over the ciliary region and therefore appears blue. The cornea is globular and opacities in it are due to ruptures in Descemet's membrane. The tension is increased but often difficult to detect clinically. The myopia is less than might be expected. This is due to flattening of the lens and its backward displacement, and flattening of the cornea.[87]

According to Foster Moore[46] there is no relationship between a high systolic pressure in the brachial artery and glaucoma. The blood pressure of fifty patients suffering from primary glaucoma, whose average age was 56, was 157 mm. which is not excessive for people this age. Conversely, the average intraocular pressure of 37 patients, all of whose blood pressure was less than 160 mm. was 14.3 mm., whereas that of 26 patients whose blood pressure in every case was greater than 220 mm., was 15.3 mm. That is, in the latter group the intraocular tension was 1 mm. higher, although the average blood pressure was higher by 78 mm.

Arteriosclerosis of the intraocular vessels does not always run parallel to arteriosclerosis elsewhere in the body. According to Foster Moore[45] of 44 patients who had a gross lesion of the cerebral arteries, 70% exhibited evidence of disease in the retinal arteries. The ophthalmoscope revealed no disease of the retinal arteries in 30% of the cases under investigation. Occasional cases[45] of sclerosed retinal arteries associated with normal blood pressure have been reported. According to O'Hare and Walker,[51] sixteen case reports of this type have been collected. They claim that these individuals at one time had high blood pressure which has since subsided. Their sixteen cases revealed a myocardial weakening or a loss of vascular tone.

Foster Moore[47] found that glaucoma[49] supervenes in a good many

cases of thrombosis of the central retinal vein. Coats[48] claims that the venous obstruction is due to thrombosis.

Verhoeff[48] maintains that the venous obstruction is due to a process of obliterative endophlebitis—a thickening of the endothelial tissue lining the lumen of the affected vein. Allen Greenwood[52] believes that this is brought on by pressure of a thickened artery crossing a vein.

Within twelve months Foster Moore[50] collected among his patients at the Moorfields Eye Hospital and those of his colleagues sixty-two examples of this condition and found that central vein obstruction causes acute glaucoma in a few weeks after onset. In other cases a slow degeneration from chronic glaucoma occurs, the disc becoming deeply excavated.

According to Coats[50] (Royal London Ophthalmic Hospital Reports, 1910, XVIII, 33) the average interval between the thrombosis and the onset of glaucoma was 100 days, eighteen out of twenty occurring within 15 weeks, the longest interval being 40 weeks. Of Foster Moore's cases,[50] the average interval was eighty days, the shortest 27 days, and the longest 210 days.

In many recovered cases, bunches of tortuous veins are developed on the disc, the result of development of collateral channels[50] between the choroidal and retinal systems. In other recovered cases circulation is restored by a process which Coats describes as a canalisation of the clot.[47]

It is my opinion that edema of the intraocular tissues, apart from the rest of the body is not a primary factor in the causation of glaucoma. Evidence points definitely to the fact that edema is secondary to increased intraocular pressure, which is caused by dilation of the intraocular capillaries, arterioles or both which retards the venous outflow of blood. According to Evans, the main factor emphasized in the production of defects brought on by edema is venous stasis.[63]

Edema of the eyeballs resulting in chronic glaucoma was observed in cases of epidemic dropsy. This disease occurred in the region of Calcutta, India, in 1909. A considerable portion of those affected developed chronic glaucoma. (Mitra, Calcutta Medical Journal, 1910-41), (Ghosh, Calcutta Medical Journal, 1910, 121).[48]

Maynard[48] in the Indian Medical Gazette, 1921, reports 104 cases, 80 in males and 24 in females. The average age of the patients was 34, contrasting markedly with that in primary glaucoma. The average time

of onset after the beginning of the disease was five months. *It is to be noted that progress of the glaucoma was arrested spontaneously although no treatment had been instituted for the greater part of the time over a period of two years.*

Since most cases of glaucoma occur in later years, some may argue that arteriosclerosis of the choroidal vessels cannot be considered as a factor in the prevention or relief of primary glaucoma.

While a sclerosed retinal artery does not necessarily produce a venous obstruction when crossing a retinal vein, venous outflow may be markedly curtailed by the pressure of a crossing hardened and thickened retinal artery. This is observed with the ophthalmoscope. The extent of retinal venous retardation so caused will depend on the extent of arteriosclerosis and the intraocular pressure, the greater the pressure, the more marked will be the venous obstruction. Exposure to darkness will cause further increase of tension, causing more compression on the crossed retinal vein.

Since arteries and veins of the retina are terminal vessels normally devoid of anastomoses, we can readily see how obstruction of a central retinal vein can cause primary glaucoma.

When the outflow of venous blood through the venae vorticosae is impeded in glaucoma, Fuchs[39] has shown that we see the anterior ciliary veins taking their place and carrying off larger quantities of blood than usual.

According to Gradle, in the beginning of simple or compensated glaucoma, the increase in pressure may be so slight that only repeated measurements at various times of the day will reveal pressure increases. As the disease progresses, the amount of hypertension increases, and the difference between the early morning high and the afternoon low becomes less.[41] After glaucoma has been in existence for some time, the chances of relief by exposure to light or any other therapeutic means are diminished because of extreme edema of the ciliary body and ciliary processes.[80] In the histological study of all of Friedenwald's[80] cases of acute glaucoma, edema of the ciliary body and processes was sufficient to account for the shallowness of the anterior chamber and the obstruction of the filtration angle. Hemorrhagic, serous or fibrinous extravasations in the ciliary processes were seen in the earlier and more acute cases, the lesion being most marked in the anterior portions of the ciliary

processes, which were swollen to two or three times their normal thickness. The presence of fibrin about the capillaries, according to Friedenwald, indicated a leakage of plasma out of the vessels, with subsequent clotting in the perivascular spaces. There was also an edema of the cornea, iris and choroid.[80]

Wolff[80] claims that acute glaucoma is associated with a vascular lesion which is localized most prominently in the capillaries of the ciliary body and processes, and that such an injury to the capillary walls with hemorrhagic, serous and fibrinous extravasations could result from any one of three causes:

1. Venous stasis.

2. Vasomotor disturbances.

3. The presence of some irritant substance of the nature of a capillary poison in the tissues.

1. *Venous stasis* may be regarded as a cause secondary to dilation of the choroidal capillaries. As Wolff claims, increased tension first acts on the venae vorticosae[81] which traverse the sclera obliquely. As a result of this, the anterior ciliary veins have to take on their work and become distended.[39,81] Later, the lumen of the venae vorticosae is narrowed because of endothelial proliferation. According to Heerfordt,[81] this is the cause of the intraocular stasis. Obstruction of a central retinal vein is secondary to pressure of a crossing thickened and sclerosed retinal artery. In the latter case, the intraocular tension[52] will determine the amount of pressure exerted on a retinal vein by a thickened and sclerosed retinal artery.

2. *Vasomotor disturbances* causing the pathologic increase in tension arise according to Adler[38] in the vessels of the chorio-capillaris. According to Wolff[39] the vessels of the chorio-capillaris become wider anteriorly, being remarkably wide at the ora serrata. The pressure resulting from the very marked dilation of these anterior choroidal capillaries may account for the extensive conjestion of the ciliary body and processes.

3. The possibility of the presence of some irritant substance of the nature of a capillary poison as the cause of the injury to the capillary walls of the ciliary body, hemorrhages, serous and fibrinous extravasation, does not seem likely. Since the posterior ciliary arteries feed the choroid and ciliary body,[84] the presence of a capillary poison would affect the vessels of the chorio-capillaris as well as those of the ciliary body. While

there is an edema of the iris and choroid,[80] there are no hemorrhages or extravasations of the blood vessels of the latter organs. The extravasations found in the ciliary body are due, I believe, to its anatomic structure. As was shown previously, the histologic study of all of Friedenwald's cases of acute glaucoma reveal that the extravasations were most marked in the anterior portion of the ciliary processes, being swollen two or three times their normal thickness.[80] According to Wolff [85] the lamina fusca resembles that of the choroid in its posterior part, but anteriorly, according to Rochon Duvigneaud,[85] is more of a serous space. According to Wolff [85] this is due to the fact that the lamellae and muscle stars gradually lose themselves in the ciliary muscle, so that beyond the middle of the muscle the space usually contains neither of these. Secondly the ciliary processes consist essentially of blood vessels, for the most part veins.[85] According to Weber, [94] swelling of the ciliary processes is due to venous stasis. Another reason may be that the walls of the capillaries of the ciliary processes may have more highly filtrative cells.[97]

During senility, according to Salzmann,[98] there is an enlargement and thickening of the ciliary processes, and according to Priestley-Smith,[99] there is an enlargement of the lens as age advances. Both of these factors, according to Salzmann,[98] account for the lessened depth of the anterior chamber and a narrowing of the free space of the posterior chamber.

I do not believe these factors account for the more frequent occurrence of primary glaucoma in old age because statistics show that age or the refractive condition have no effect on the intraocular pressure.[1] Furthermore, Salzmann has found [98] that enlargement of the lens and ciliary processes during senility is compensated by the defective secretion of aqueous. If the volume of the eyeball were increased in old age, the average intraocular tension during senility would be higher than in younger years. Besides Parsons [23] has shown that attacks of acute glaucoma occur especially at night, and Alger [33] has shown that cases of chronic simple glaucoma reveal abnormally high tension only during the night.

Friedenwald [80] believes that a histamine-like substance is responsible for the vascular crisis which brings about the attack of acute glaucoma. While he has shown that aqueous from a case of acute glaucoma contained a toxic substance which raised a small wheal when injected under the skin, it must be remembered that the high protein content of the aqueous in glaucoma is apt to cause a local allergic reaction in one hypersensitive to such a protein. Proteins from other sources, when in-

jected hypodermically, are known to cause a wheal in those hypersensitive to the particular protein used. The size of the wheal depends on the extent to which the subject may be hypersensitive. This toxic substance in the aqueous of a glaucomatous eye is, in my opinion the result rather than the cause of primary glaucoma. Furthermore there is every reason to believe that the presence of a toxic substance in the aqueous would cause a hyperemia and swelling of the iris, as well as a cloudiness of the aqueous some time before an acute attack of primary glaucoma; *yet these symptoms are not known to appear before attacks of acute glaucoma.*

Friedenwald,[95] like Duke-Elder,[4] believes that dilation of the intraocular vessels increases their permeability, thereby increasing the dialyzed fluid content of the eyeball. Gradle[7] has shown that of glaucomatous patients kept in a very dark room for one hour, more than 50% will show the tonometric findings at the end of this period to be increased from 15 to 40 mm. Hg. above the reading taken before being placed in the dark room, and upon exposure to strong light, there follows a *sudden* drop in tension to the *previous* level. This fact appears to prove that it is the actual dilation of the intraocular vessels and not the increased dialyzed fluid content (thought to be caused by the increased permeability of the dilated vessel walls) that causes the increased pressure. If the increased tension were actually due even partly to an increased dialyzed fluid content of the eyeball, exposure to light would cause only a partial drop in pressure.

It does not seem likely that a histamine-like substance is responsible for the vascular crisis which brings about an attack of acute glaucoma as postulated by Friedenwald.[80] While he found it possible[95] to produce a rise in intraocular pressure in experimental animals by injecting histamine into the vitreous, it will be noted that the rise in pressure was of short duration and *was followed by a very marked fall in pressure. This is not at all characteristic of attacks of acute glaucoma.*

In the experimental animals in whom histamine had been injected into the vitreous, the ciliary bodies were found to be edematous, resembling somewhat the condition found in acute glaucoma in human eyes.[95] Since the histamine caused a rise in pressure which was of short duration, followed by a very marked fall in pressure, it appears very evident that the rise in pressure is not due to edema of the ciliary processes. This seems to show that the primary factor causing the increased pressure is the dilation of the choroidal capillaries. Friedenwald[95] admits this is so.

Since the ciliary processes consist mostly of veins,[85] it appears that edema of the ciliary body is caused by venous stasis[94] which is secondary to pressure changes caused by dilation of the choroidal capillaries.

It must be remembered that we are concerned primarily with prevention—that is, avoidance of darkness at all times, even in the very early stages of primary glaucoma. Those cases where the patient is awakened from sleep during the night or the very early morning with ocular and head pains, nausea, vomiting, or difficult breathing, are the cases to be suspected of preglaucoma.

A patient of mine, a married woman in her thirties, came to me with a history of severe ocular and head pains, accompanied by vomiting about two or three A. M. every morning for a period of about seven years. Repeated physical examinations by various physicians revealed no abnormality. There was no refractive error found. I advised this patient to sleep in a lighted room, but no promise of relief was made. She told me that her condition caused her to be so nervous that she was afraid to sleep in the dark. About two years previous to her visit to me she had an oil lamp burning all night in her bed room for a period of about two months, during which time her symptoms disappeared entirely. Having no longer felt nervous, she discontinued sleeping with a light in her bed room, with the result that her symptoms returned immediately. She reported back two days later after her first visit to me with the result that her symptoms were markedly relieved. She used a seven watt bulb placed about ten feet from her face. I advised her to keep the light no further than two or three feet from her face and then report back to me if this caused any further relief. Lo and behold, this caused complete relief! Ever since instituting this preventive therapy, she is now able to eat before going to sleep. During the period of her suffering, her husband resorted to frequent bouts of drinking for obvious reasons. She reported recently that my advice has resulted in a happy married life once more. I am making no attempt to dramatize this point. I desire only to emphasize the extreme importance of the use of light in a case such as this.

If sleeping with a light over the face relieves the symptoms or prevents further occurrences of being awakened from sleep with the above symptoms, we are justified in assuming that we are dealing with a preglaucomatous condition.

These prophylactic measures are justified because we have definite

evidence that darkness causes hypertension, particularly in those subject to or having glaucoma.[5,6,7,23,33]

Recently [88] a case of glaucoma at the Royal Westminster Ophthalmic Hospital was reported to be cured without operation by short-wave therapy.

I have found that when those patients who are awakened from sleep with the above symptoms, go to sleep earlier, they are either awakened from sleep at an earlier hour or the symptoms are more severe; and when going to bed very late, that is about 1 A. M. or later, the symptoms would not manifest themselves until about 5 or 6 A. M. This point emphasizes the fact that it is exposure to darkness that appears to cause the above symptoms. It also seems evident that the hour of maximum intraocular pressure will depend on the time the patient goes to sleep, and the phenomena associated with the individual's dark adaptive properties of the retina, for Gradle [7] has shown that of glaucomatous patients kept in a very dark room for one hour, more than 50% will show the tonometric findings at the end of this period to be increased from 15 to 40 mm. Hg. above the reading taken before being placed in the dark room.

Two of my patients with a cardiac involvement reported a choking feeling when sleeping in a dark room. The use of a lighted seven watt bulb over the face when sleeping gave complete relief. In one case the fuse blew out during sleep two different nights. The patient was awakened from sleep each time with a choking feeling. This fact rules out any psychologic influences.

It is interesting to note that many heart attacks occur at night during sleep or in the very early morning hours before arising.

Luckiesh and Moss [18] have shown that reading under 100 foot candles in normal healthy subjects causes a slowing of the heart rate to the extent of 2%, and under 1 foot candle the heart rate is slowed down to the extent of 10%. Parsons has shown that attacks of acute glaucoma occur especially at night,[23] and Alger has shown that after the onset of presbyopia in middle life, chronic simple glaucoma reveals abnormally high tension only during the night.[33] Gradle [7] has shown that of glaucomatous patients submitted to the dark room test, over 50% revealed that the tonometric findings at the end of one hour were increased from 15 to 40 mm. Hg. above the reading taken before being placed in the

dark room. The statistical pressure[1] in normal or glaucomatous eyes is greatest in the early morning hours.

It is to be specifically noted that many heart attacks occur during the same period of time when the intraocular tension is at its peak. It is my conclusion therefore that the oculo cardiac reflex[19] is the cause of heart attacks occurring during the night or the early morning hours before arising, for Adler[19] has shown that through the oculocardiac reflex, pressure in or on the eyeball causes a slowing of the heart rate. Increased intraocular tension, as was previously brought out, may give rise to nausea and vomiting via the oculocardiac reflex. Parsons[23] has shown that acute attacks of glaucoma cause vomiting, and the attack is liable to be mistaken for a severe "bilious attack." He stated further that the constitutional disturbance is often so great that the patient is prostrated, and the pulse is irregular and intermittent.

It appears therefore that darkness acts as a cardiac depressant.

ON THE ETIOLOGY OF PRIMARY GLAUCOMA

The etiology of primary glaucoma appears to originate in the chemical anomalies of the regenerated visual purple in darkness.

Exposure to light causes the retinal tissues to become acid in reaction,[53] the degree of acidity varying directly with the intensity and duration of the exposure and the wave length, yellow-green light being most effective. The production of acid occurs more rapidly when the retina is in contact with the pigment epithelium than in the isolated retina. It is thought that the development of acidity in the retina may prevent the reformation of the visual purple during exposure to light.[53] Regeneration of the visual purple occurs only if contact with the retinal epithelium be retained.[53]

According to Adler,[54] after contusions of the globe which do not rupture the external coats, hypotension is commonly seen, and detachment of the retina is nearly always followed by a period of hypotension. Würdemann[55] has observed that in cases of traumatic detachment of the retina the anterior chamber is often strikingly deep and the tension is diminished. In detachment of the retina, the pigment epithelium always adheres to the choroid.[56]

What appears clinically as a retinal detachment is, in the large majority of cases, not a true detachment, but a separation of the nervous portion from the pigmentary layer.[69] It consists actually of the reforma-

tion of the cavity of the primary optic vescicle. According to Wolff the only true detachment of the retina is in cases of sarcoma of the choroid. [69]

The hypotension that follows contusions of the globe which do not rupture the external coats, is brought on, I believe, by a chemical disturbance of the retinal elements. This is shown in those cases where we see stars when receiving a sufficiently jarring blow to the jaw or head, or when being poked in the eye with a blunt object. Such a mechanical disturbance may give rise to a chemical imbalance of the rods, probably affecting the chemical character, the quantity of formation, or both of the visual purple, resulting in constriction of the choroidal capillaries. This appears to be evident because retinal detachment sometimes follows. *It therefore seems that hypotension precedes the retinal detachment.* Nothing else but a marked constriction of the choroidal vessels, exclusive of a subretinal hemorrhage or edema can account for a detachment of the unruptured retina in contusions of the eyeball where the external coats have not been ruptured.

Oguchi's disease,[90] a congenital and stationary form of night blindness first described by Oguchi (1907) in Japan from which country most cases have been recorded, appears to show that the respective bleaching and regeneration of the visual purple causes the constriction and dilation of the choroidal vessels. Ordinarily, in this disease, the posterior region, and sometimes the entire fundus is of a grey color, which is probably due to a layer of abnormal tissue between the cones and the pigment epithelium. After some hours in the dark, the grey color is replaced by the normal red appearance. In dim illumination, night blindness exists until several hours have been spent in the dark, when vision slowly improves. It is to be noted that after some hours in the dark, the fundus assumes a normal red color simultaneously with the improvement of dark adaptation. It seems likely that the regeneration of the visual purple is closely related to the dilation of the choroidal vessels which is made evident by the fact that the fundus assumes a normal red color after remaining in the dark a few hours. This demonstrates that darkness causes dilation of the intraocular vessels resulting in increased vascularization of the inner eyeball.[5,6,7,23,33,38]

The reformation of visual purple takes about one hour.[57] Gradle[7] has shown that in more than 50% of glaucomatous patients submitted to the dark room test for one hour, the tonometric findings were found to be from 15 to 40 mm. Hg. above the reading taken before being placed

in the dark room. This appears to show the close relationship between the intraocular pressure and the regeneration of the visual purple.

According to Waite, Derby and Kirk,[58] the rate of dark adaptation is invariably retarded in the glaucomatous eye, even in the early stages of the disease. The fact that exposure to darkness causes a pathologic increase in tension in those subject to glaucoma, and the fact that the rate of dark adaptation is retarded seems to show the anomalous chemical character of the regenerated visual purple.

According to Hecht (1919-25)[61] the visual purple is a photochemical substance split up under the influence of light into two components, and that when the light is removed, these two components combine again to reform the original substance.

Since poor dark adaptation is found in eyes having hyper as well as normal tension (such as cases of retinal pigmentary degeneration),[59,102] it seems that these two components forming the visual purple do not combine completely with the result that some of each component is free to act individually, one affecting dark adaptation and the other causing dilation of the intraocular vessels, both substances being effective only in the absence of light, since we know that the intraocular vessels dilate only in darkness.[5,7,23,33,90] This latter fact is well demonstrated in Oguchi's disease,[90] where the grey color of the fundus is replaced by the normal red appearance after remaining in the dark a few hours.

To show further that the action of the visual purple appears to be the interposing mechanism controlling the constriction and dilation of the choroidal vessels, we have only to consider the direct action of light on blood vessels in the skin, where we have the opposite effect caused by light and darkness.

I shall quote from Laurens: "Light, within physiological limits, may cause the cutaneous vessels to dilate and thereby allow more blood to go to the skin. During long continued exclusion of light the skin vessels become constricted and the skin thus assumes a bloodless appearance."[75]

Quote: "Balderry and Barkus (1924) demonstrated that the primary effect of irradiation is a vasodilation."[76]

We therefore see that whereas light causes constriction of the choroidal vessels, it causes dilation of the cutaneous vessels; and darkness

causes dilation of the choroidal vessels and constriction of the cutaneous vessels.

To show further that the visual purple appears to be the interposing mechanism between light differences and the constriction and dilation of the choroidal capillaries, Stallard [103] has shown that the prognosis of a detached choroid is favorable if the overlying retina is not detached.

In eyes blind from glaucoma light and darkness will cause little if any further pressure changes because Wolff [72] has shown that the smaller vessels of the choroid, especially the capillaries, are occluded and tend to disappear.

According to Wolff the pigment epithelium remains intact a long time, and the rods and cones may remain intact in eyes blind from glaucoma.[73]

If it were not for the occlusion of the smaller choroidal vessels in eyes blind from glaucoma, it may be likely that light and darkness would cause variations of the intraocular tension because Feigenbaum (1928-29) [5] has shown that in dark adaptation the intraocular vessels dilate and that the tension rises in blind as well as seeing eyes.

If the foregoing statements be true, then it appears that the physiology of the liver may have some relationship with the intraocular pressure. In the frog, pigment migration is stopped by the subcutaneous injection of bile salts (Yamada, 1919),[65] or the isolation of the liver from the general circulation. (Kumagai, 1915.)[65]

Pathological changes in the liver have been associated with the etiology of night blindness. Injections of bile and bile derivatives have a similar result in experimental animals.[66] According to Percy W. Cobb, in diseases of the liver, night blindness is closely associated with jaundice.[77] According to Adler,[78] there is clinical evidence that some substance present in the liver is concerned with rod-visual purple function.

CONCLUSION:

The causes of primary glaucoma may be:

1. Obstruction of a central retinal vein.

2. Anomalous behavior of the visual purple which seems to be responsible for the dilation of the intraocular vessels when exposed to darkness, resulting in increased intraocular tension.

It appears that nocturnal and early morning heart attacks occur when the intraocular tension is at its peak. The hour of occurrence depends on the time that the patient retires. When retiring earlier, the rise in intraocular tension with its attendant symptoms appear earlier, unless the patient sleeps in a lighted room.

It appears that the destructive forces of primary glaucoma exert their telling effects such as glaucomatous cupping of the disc, deterioration of vision, etc., during the time when exposed to darkness because:

1. Parsons[23] has shown that attacks of acute glaucoma occur especially at night.

2. Alger[33] has shown that after the onset of presbyopia in middle life, chronic simple glaucoma reveals abnormally high tension only during the night.

3. Gradle[7] has shown that of glaucomatous patients kept in a very dark room for one hour, more than 50% will show the tonometric findings at the end of this period to be increased from 15 to 40 mm. Hg. above the reading taken before being placed in the dark room. Upon exposure to strong light, there followed a *sudden drop* in tension to the *previous level.*

From cases observed in practice, the time of appearance of preglaucomatous or glaucomatous symptoms depends on the time the patient retires, that is, when retiring earlier, the symptoms appear sooner unless the patient sleeps in a lighted room.

From cases observed in practice, primary glaucoma is preceded by a stage of preglaucoma which may be of a few years duration. It may be from three to seven years before full blown acute attacks of primary glaucoma occur.

The only reliable diagnosis appears to be based on the case history.

The *ocular* origin of primary glaucoma appears to rest entirely in the retina, that is, in its circulatory and visual purple forming mechanism, as well as the anomalous behavior of the visual purple itself.

Since Feigenbaum (1928-29)[5,6] has shown that dark adaptation causes the intraocular pressure to rise in cases of aniridia, after an iridectomy, and with pilocarpine, it seems evident that light and darkness supersede the action of drugs and surgery.

The prevention and control of primary glaucoma that is not due to obstruction of a central retinal vein therefore *rests in the avoidance of darkness at all times.*

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PUBLIC HEALTH*

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The 42nd Annual Congress and Convention of the American Optometric Association was opened at Los Angeles, June 25th, by the First National Conference of Optometrists.

This conference was a refreshing indication that the American optometrist has at last emerged from the welter of confusion and uncertainty that surrounded the question of "Public Health" and his relation to it—a question considerably muddled, purposely or inadvertently, by the loose use of such terms as "socialized medicine" or "governmental interference" and poorly founded attacks on a National Health Program supported in part by the federal government.

In a recent editorial in the AMERICAL JOURNAL OF OPTOMETRY, Health Insurance was indicted on three major counts: (1) cost, (2), "governmental control," and (3) the fact that the American Medical Association doesn't like it.

The Wagner National Health Bill (introduced in Congress following President Roosevelt's message submitting the recommendations of the National Health Conference) is not primarily concerned with Health Insurance. The bill places greatest emphasis on child welfare. It is left to the respective states to elect whether they wish to provide for child, maternal, rural or indigent health services.

No state is compelled to adopt any sort of Health Insurance Plan. It would be well, however, for the optometrist to note closely that medical societies in various states are now sponsoring independent bills which will provide for payment of medical fees through insurance.

In view of these facts, it is imperative that the optometrist clearly understand his relation to "Public Health."

*Submitted for publication June 29, 1939.

"Public Health" includes eye care in hospitals, schools, health centers, industrial plans, group plans, clinics, and for relief and WPA patients, etc. These services are steadily and inexorably expanding, not contracting, and optometrists can only find their rightful place in them through the medium of a constructive program.

Perhaps no other single factor has contributed more toward confusing the issue than the indiscriminate use of the term "socialized medicine."

"Socialized medicine" is not the issue—even if two American professional men can be found who agree on what "socialized medicine" and "socialized medical service" really mean. Some practitioners support the extension of health services to every person requiring such services. Others wish to limit supplying health services to the indigent and still others are opposed to any extension of health services whatsoever.

The editorial admits that "there is a serious need for an extension of health services on some basis that will enable all groups to possess them at a cost that is not too burdensome."

The fact is that the entire country is moving in a powerful, though slow and steady, current toward the proposition that adequate health facilities must be made available to all three-thirds of the American population.

To deny the existence of that current or to attempt to stem the tide could only have in the writer's opinion a disastrous result for optometry.

The various programs in the different states giving aid to the blind have as part of their aim the institution of measures for the prevention of blindness and the restoration of eye-sight. These plans are not projected, nebulous or remote—they are in existence and include opthalmologists but not optometrists.

These plans must be accepted as faits accompli in answer to the powerful wide-spread demand for health security.

Criticism not based on careful study of administrative problems can serve no useful end. Such criticism can only serve to alienate the administrators and agencies that have the responsibility of putting health programs into effect. It would patently be of more advantage to optometry to present ideas for improved administration and efficiency that are based

on careful study and knowledge of eye care problems.

Every administrator will welcome optometric representatives bearing constructive suggestions that would tend to increase efficiency and effectiveness of the services rendered.

With the full recognition by the optometrist that the Public Health programs are here in one form or another and are here to stay he will concentrate his efforts toward attaining a responsible, considerable and honorable position in the eye care field of public health.

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POLICIES RELATING TO PUBLIC WELFARE

In recent years there has developed a growing concern over the increasing number of optometrical organizations. While the American Optometric Association has increased its membership during the past five years, the fact remains that many optometrical organizations have been formed during this period, and in reviewing the activities of some of these, one is struck with the thought that some at least are unnecessary, and are a serious drain on the time and interest of optometrists. In this connection it is well to recall that the American Optometric Association always has assumed and always will assume the responsibility of optometric progress and the defense of all that has been accomplished for this profession in the past. No other group of optometrists is representative of all types of thinking in optometry. No other group of optometrists has the organization necessary to carry out the tasks before it and no other organization includes within its membership practically all members of all independent groups.

It is also well to recall that most of these newly formed optomet-

rical organizations were originally organized for purely scientific purposes in some particular segment of the ocular field. Yet it is a rather common occurrence to find officers of these independent organizations expressing endorsement of policies in conflict with those previously endorsed by the American Optometric Association. Policies having to do with the general welfare of optometry and policies frequently having nothing at all to do with the technical side of the work done by optometrists.

Now obviously no one would deny an officer of an independent optometric organization the American right to express his individual opinion on any matter whatsoever, yet when an individual speaks as member of the governing board of an optometric organization, on a matter of importance dealing with broad policies to be followed by all optometrists we believe it is fair to at least ask the question, "Is he speaking in the name of his organization?" It is equally obvious that an officer of the American Optometric Association should not and may not voice policies which have not been approved by that organization and it must be remembered that in the public mind an officer of an optometric organization can not be disassociated from his official position with his organization.

From the above, two thoughts stand forth. First, that on matters of broad public policy it should be the responsibility of the American Optometric Association to speak for the profession, and Second that officers of all optometrical organizations during their term of office refrain from public discussion of broad policies relating to public welfare, except as such have been officially adopted by their respective organizations. The adoption of these rules of conduct will save optometry much embarrassment in the days which are to come.

CAREL C. KOCH.

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SPECIAL REPORTS

OFFICIAL VIEWS OF THE AMERICAN MEDICAL ASSOCIATION REGARDING THE WAGNER HEALTH BILL NOW BEFORE CONGRESS*

Official representatives of the American Medical Association appeared before the Committee on Education and Labor of the United

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States Senate on May 25. The committee was conducting hearings on the proposed Wagner Health Bill, (S.1620). The following statements were presented regarding the attitude of the American Medical Association towards the proposed legislation.

Dr. R. G. Leland, director of the Bureau of Medical Economics of the American Medical Association:

"The bill now pending before your committee is obviously based on the report of the Interdepartmental Committee, and that report in turn is based on the report of its Technical Committee. The report of the Technical Committee seems to be based on figures assembled by that committee that the committee itself seems to regard as supporting the thesis that death and sickness rates in the United States are excessive; that this excess depends not on economic conditions but on inadequate medical service, and that by expending a vast sum of money under federal supervision the sickness and the mortality rates can be substantially reduced. The figures relied on by the Technical Committee to support its thesis are believed to be incomplete and the deductions drawn from them are believed to be unwarranted by any available evidence. The conclusion that the reduction of sickness and mortality rates awaits only large federal and state appropriations, expended under federal supervision and control, is believed to be without foundation . . ."

Dr. R. L. Sensenich, South Bend, Ind., member of the Board of Trustees of the American Medical Association:

" . . . the federal government should have no authority to dominate the policies or personnel of the hospitals or to make working agreements binding the community directly or indirectly in the operation of its hospitals . . .

"From the definition of the term hospital as used in the title, 'includes health, diagnostic and treament centers, institutions and related facilities,' it is evident that more is contemplated under the proposed legislation than is ordinarily included under the term of hospitals. Otherwise there would be no reason for introducing this vague and all inclusive definition. It is noted that the Technical Committee in its report proposed the construction of 500 health and diagnostic clinics in areas in which the individual does not have convenient access to hospitals.

"A plan for the establishment of clinics for the diagnosis and treatment of ambulant patients in areas already supplied with physicians should be weighed carefully from the standpoint of the ultimate best medical service to the community as a whole. If the clinic contemplated would encourage the patient to present himself directly to the clinic for examination and treatment, it would not offer anything superior to dispensary service. The latter type of service lacks needed medical history of other illnesses and treatment, and opportunity for observation. This tends to detachment of interest, and the responsibility of the medical staff member to the patient is soon replaced by responsibility only to the system which employs him. There is reason to believe that the service would not be superior to that offered by physicians in the community.

"Such a clinic is an outgrowth of the view entertained by some that Medicine can be 'depersonalized'—that the physical man can be treated without intimate knowledge as to his emotional stresses and the effects of his contact with his environment.

"The establishment of clinics for treatment of ambulant patients only assumes further that the individual may have one physician for illnesses in which he is ambulant and another when he is confined, without loss of values which are of importance in the best medical service. Obviously these are faulty assumptions.

"It must be recognized that the private physician will still be necessary to attend patients who are not able to go to the clinic. The well trained private physician, with a closer and more personalized service to the individual and his family in their home, will give good medical service to more people. He should be given every opportunity and assistance to increase his usefulness, and governmental machinery should not be

permitted to impair it. The operation of large diagnostic and treatment clinics recently established in very populous centers of impoverished people cannot be fully evaluated on so brief experience. Obviously, it cannot be translated into terms of small rural areas."

Dr. Haven Emerson, professor of public health practice, College of Physicians and Surgeons of Columbia University, New York City:

"The record of progressively improving health in the United States of America for the past fifty years and particularly for the past eleven years impresses me with the trustworthiness of the official and voluntary agencies and institutions and methods of local, county and city and state government developed by long experience and appropriate adjustment for prevention of disease and care of the sick.

"No emergency of sickness or neglect faces us. No spectacular or immediate improvement in general health can be expected through extensive changes in procedures, in responsibility, or in sources of funds as proposed in the Wagner bill.

"Better national health can be assured by development along well established lines in the orderly evolution of social and professional resources made available by new medical knowledge.

"Better care of the sick, and more nearly universal availability of the increasingly costly facilities for diagnosis and treatment, can be assured by extension of the various promising experiments such as voluntary prepayment and cash indemnity plans than can be expected from attempts to have the cost of sickness transferred to the federal government or by relying on state and local health officers to administer treatment facilities at public expense.

"We, as physicians, whether engaged in individual practice of curative medicine or as officers of health concerned with the administrative application of preventive medicine for local and state governments, advise against disturbing a series of services which have a long history of success, by creation of another policy where local responsibility will be largely at the mercy of federal dominance and allocation of funds.

"Estimates of neglected sickness have been much exaggerated. Theoretically possible benefits from large additional expenditures are too optimistic and problematic to be convincing.

"As the major cause of insufficient care for the sick and for health, where these exist, is lack of earning power and the dislocation of commerce, industry, production and demand for goods, characteristic of the past eleven years, so it will be the reestablishment of sound economic and employment conditions which will correct the error, rather than large spending programs by government which do not lead to any basic improvement in the incomes of wage earners. Neglected sickness is less often the cause of economic misfortunes than vice versa . . .

"It is wholly undesirable to have any state or federal plan for care of the sick depend on the leadership, professional direction or financial control of the state or local health officers. Those that are competent are more than fully occupied preventing and controlling disease and directing sanitary measures for health. Those that are not specialists in health are even less qualified as experts in the intricacies of services for the sick . . .

". . . I do not believe it is the function of the state or national government to take care of women in confinement or to take care of babies or to take care of other things which are the functions of the practice of medicine and which can be better handled by local communities than it can by aid from Washington. I believe that the most intelligent expenditure of what you might call stimulating money for the health of the nation would be in the field of prevention rather than in the field of the care of the sick. I think the ordinary instincts of the United States are sufficiently developed so that whenever it is called to our attention that there are neglected sick people nearby in the counties and states, that you will find provision made to take care of them. But it takes a great deal of imagination and a great deal of statesmanship to spend for the preventive mechanism. The people do not see that close enough. I believe that it is

the proper functioning of the federal government to assist the states that cannot do it themselves to have a really well developed, a well rounded full-time public health service based on a county and state organization. That would seem to me to be the first object and the only really immediate object for which this expenditure might be asked."

* * * * *

OPTOMETRIC REFLECTIONS*

Walter Gasson, F.B.O.A., F.M.S.C.
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Aren't optometrists a rum lot? They are complacent and yet critical of other people's faults. Like Mr. Micawber, the immortal Dickensian character, we are waiting for something to turn up without troubling about it, and at the same time believing *Nil Desperandum*. We are, generally speaking, born grumblers. Listen to travelers' tales! After all, the traveler should know as they meet so many of us up and down the country on their rounds. We must be a miserable lot of specimens.

Why cannot we develop a little more enthusiasm in our work. Perhaps the fault lies in ourselves and not as Shakespeare says "in our stars." Pessimism is a chronic complaint which should never be allowed to gain the upper hand. All of us have our bad patches and after a few years' experience we can generally tell when and when not to expect them. It seems as though we are all too ready to judge ourselves by our failures and not by our successes—and we all have both.

Oscar Wilde neatly summed up the situation in the celebrated epigram, "Experience is the name one gives to their mistakes." The best of us can be guilty of occasional lapses. It's a human trait and we would be something other than human if we did not occasionally slip up. Have we never had several weeks of satisfactory transactions and then for a week or so nothing else but readjustments, alterations and grumbles? We seem to judge ourselves by this occasional week and not by the several weeks of self-pleasure giving satisfaction.

Optometrists as a class may lack a little of the appreciation of the other man's point of view. The understanding of the other side of the question does so much more in the satisfying of a critical and perhaps hostile patient even when we know full well that it is not ourselves who are unreasonable. On the other hand, there does come a time when it is

*Submitted for publication March 14, 1939.

inadvisable to give way and it is the man who knows his job who can judge the right time to stand up for himself.

We tend to look upon a refraction case as another in helping to make up the week's work. Yet to our patient this is an event in their lives by which they can associate corresponding occurrences. To have made up their minds to have their eyes examined may have taken weeks of self-persuasion by themselves, their family and friends.

The optometrist who puts service before self as his motto and goes out of his way to command the appreciation and perhaps the respect of his patient is the successful optometrist. If we are fortunate in creating a favorable impression we may look forward to future recommendations. The happy and satisfied patient is almost a friend for life.

We can only do our work to the best of our ability when we understand our patient, his faults, failings and character. There are generally speaking, three individualistic types of patient with which we may have to deal. The first who demands and expects the very best and is willing to pay for this service. The second, the complacent type, who leaves the outcome of the refraction in the optometrists' hands, and is probably the nicest type of patient with whom to deal. We have the third who do not appreciate the value of their eyes in the proper way and expect and demand the cheapest of the cheap. The successful optometrist can correctly assess and judge his patients and deal accordingly with each of these three arbitray categories.

The science of refraction is the correct understanding of the patient's visual difficulties and the intelligent mental indexing of all their symptoms. Our patients visit us because there is something wrong or rather something in their opinion is not as it should be with their eyes. Ophthalmic optics is a livelihood which is full of interest because our patients are drawn from all classes of human society. We cannot treat any two alike. To some we must be courteous, aloof and strictly professional. Others, especially the poorer classes, we must be more understanding and try to come down a little towards their level. The understanding of the patient and his troubles, however irrelevant, insignificant or trivial, solves a greater part of the problem of refraction and its results. Our clients soon assess us and value our ability accordingly. They value our slightest remarks, however unimportant they may have been intended.

Because the general public are the judges of our ability and our

work, it is therefore preferable to adopt a professional tone. The less of a trade atmosphere we maintain the better it will be all round. It is advisable for all well-meaning optometrists who think of their future to adopt this professional attitude, but not beyond exaggerated limits. The work of the qualified optometrist is the physiological refractive care and treatment of the eyes.

Reputation is a necessary achievement for which individual optometrists must strive. This important attribute depends upon several things of which the most essential are personality, skill, education, experience and character to mention a few. The development of all, subscribe to the enchancement of a good reputation. A good name travels slower than a bad one. Of all of them, personality is probably as important as any, as personality and professionalism go together. What is more important still is to encourage the public to know that the modern qualified optometrist is no longer the seller of spectacles but a trained man in whom confidence in all ocular matters can be given.

The optometric profession has suffered two fold from increased competition within the ranks, and also unfair hostility from external sources. Success always encourages competition and only failure discourages opposition. The profession has always been comparatively successful and hence if I may coin a word "un-optical" competition has arisen. In spite of this I feel on the whole the qualified optometrist is generally speaking not very much the loser. Anyway, fair competition is an excellent thing in itself as it tends to bring out the very best qualities in the competitors. It is only when unfair means are resorted to in the form of deceit and unethical practice and undue influencing by interested parties, will the consulting optometrist have to fight for his profession. If he does so with the zeal of his work, personal contact with his patients and they in turn recognizing in him that he is the man for their ocular needs as far as refraction is concerned, the optometrist should have little to fear.

The optometric profession still largely enjoys the public's confidence and this will take a lot to undermine. If the qualified man remembers the heritage of his work and that of his predecessors who have made his calling what it is, he can face fair and unfair opposition with equanimity. We perform a public service that is in demand and we have the training and what is more the complete facilities with which to meet

the demand in a satsifactory and unequalled manner.

WALTER GASSON, F.B.O.A., F.S.M.C.
111 THE PARADE
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WATFORD, HERTS, ENGLAND

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BOOK NOTICES

THE PRACTICE OF MODERN OPTOMETRY. William Henning, Opt.D., F.A.A.O. Published by the American College of Optometrists. 1939. $10.

This new work by Henning deals with (1) a modified system of examination procedure, (2) a new system of recording refractive findings, (3) a new hypothesis as to the classification of ocular syndromes, (4) a new conception of the relationship of bodily functional disorders in relation to ocular symptoms, and (5) the use of selected visible light frequencies in certain optometrical cases. This book rather clearly presents Henning's present thinking on matters as they relate to the work of the optometrist and while many of his ideas are far from the generally accepted views he presents them in a readable manner and in one in which the reader can follow his thinking. Henning is blazing a new trail and in this work brings his readers abreast of his latest creative thinking in matters of ocular interest. Whether or not one follows Henning, his work is of interest as he is attempting to build in a field in which much work is still to be done. C.C.K.

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APPLIED ORTHOPTICS, by S. Edwin Rudlin, A.B., O.D., 194 pages with illustrations, published by the Dietz Press, Richmond, Virginia, 1939. $5.00

This handy volume, which has been placed on the recommended list at the Northern Illinois College of Optometry, belies its slender appearance in that it contains a folio of information in a concise and explicit form.

There are twelve chapters dealing with distinct phases of orthop-

tic application. The first two deal with a discussion of physiology and psychology involved, and with the setups and operation for different purposes of all commonly used instruments.

The material treated may be divided into sections. The first deals with orthoptics as applied to the strabismus case, and includes chapters upon neutralization, amblyopia, preliminary fusion, true fusion, and stereopsis. The second deals with the binocular dysfunctions, including chapters on vertical dissociation (also applicable to strabismus), accomodation-convergence, and bifocal reconditioning.

In each instance, the targets used are explained and illustrated; the procedure is outlined and explained, and application is "given" upon all makes of instruments. Home training methods are included in the discussion. Where applicable, the material is handled in both the symbols and phraseology of the Graduate Clinic Foundation, and in the standard nomenclature. Thus, both those in or without the Foundation will find the material readily comprehensible.

In the concluding chapter, the methods previously described are summarized, and in addition several techniques are offered which were developed by the author himself during his experience as head of the Orthoptic Division of the Northern Illinois College of Optometry Eye Clinic.

It must be emphasized that this book is not a treatise in exposition of some pet hobby or technique, but a rounded and inclusive discourse of the accepted and proven methods, simplified and condensed for use in actual practice. Where possible, theorizing has been eliminated, and the book shaped for ready use by the practitioner of Orthoptics.

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